ANESTHESIA-RELATED MATERNAL MORTALITY*

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Anesthesia-related maternal mortality continues to be a reality in the 1980s. To provide safe and effective anesthesia for the parturient, thorough understanding of her unique anatomy, physiology, and pharmacologic responses is required. We shall discuss the two major types of anesthesia (regional and general) commonly rendered parturient women and will attempt to clarify the factors that make the administration of anesthesia to this group of patients a discipline to be taken most seriously.

MORTALITY FROM REGIONAL ANALGESIA

Spinal shock. A decrease in the incidence of severe hypotension leading to cardiopulmonary arrest (spinal shock) in gravidae[1,2] is attributable to the recognition of two factors: first, that a pregnant woman has a decreased dose requirement for local anesthetic agents and, second, that displacement of the uterus from the abdominopelvic vessels must be combined with intravenous fluid preloading before institution of a regional block.

Reduced drug requirement. Beginning early in pregnancy and continuing to term, a smaller dose of local anesthetic drug is needed to obtain a desired level of spinal or epidural analgesia. A reduction in drug requirement averaging one third of the usual dose occurs during the final trimester and has been confirmed by three studies. Bromage,[3] investigating epidural analgesia, noted that a pregnant woman needed only 14 ml of 2% lidocaine to obtain sensory blockade to the 10th thoracic dermatome whereas a nonpregnant woman of similar height and age required 20 ml of the same drug. Comparable results were obtained with spinal analgesia. Doses of 0.2% procaine necessary to reach blockade to

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the fourth cervical dermatome were at least twice as large two days postpartum than in the same 12 women before labor.[4] And when the same dose of local anesthetic (tetracaine 5 mg–dextrose 50 mg) was injected under identical conditions to 10 parturients and 10 young gynecologic patients, the pregnant women showed a significantly faster onset, higher level, and longer duration of blockade.[5]

Several physiologic changes associated with gestation have been suggested as causes of this altered reaction to local anesthetic agents. During gestation, buffer capacity in response to the hyperventilation-induced alkalemia is diminished; as a consequence, local anesthetics remain in the ionized salt form for a longer time and therefore stay in the compartment of injection for an extended period.[6] Greater spread of local anesthetics in the subarachnoid and epidural spaces may occur as a result of distended epidural veins which reduce the capacity of both these spaces.[3,5] Furthermore, retarded venous circulation in the meninges may delay their ability to absorb local anesthetic drugs and lead to prolongation of analgesia.[5] Finally, increased concentrations of progesterone and estrogen may alter membrane permeability facilitating diffusion of the drug across the nerve sheath.[7,8]

Mechanical factors such as aortocaval compression do not seem to contribute to this altered drug requirement. First, the decrease persists despite uterine displacement or maintenance of the gravida in a lateral position.[5] Second, the reduction has been demonstrated as early as the first trimester[6] before aortocaval compression comes into play and does not end suddenly at parturition but wears off gradually over the initial two postpartum days.[9]

Intravenous fluid preloading. Subarachnoid or epidural placement of local anesthetic drugs results in sympathetic blockade with consequent postarteriolar pooling of blood and decreased circulating blood volume. In the gravida, vascular tone is more dependent upon sympathetic tone than in nonpregnant subjects,[4] and venous return to the heart is further impaired by aortocaval compression. These two factors account for the increased incidence of arterial hypotension in pregnant women during techniques of regional analgesia. However, significant falls in blood pressure following spinal or epidural analgesia in parturients can be prevented by intravenous fluid preloading associated with adequate displacement of the uterus off the great abdominopelvic vessels. Uterine displacement, which should be to the left in most gravidae, is achieved

easily by mechanical elevation of the right hip using a wedge or sandbag combined with leftward tilt of the table.

The hemodynamic benefits of fluid preloading with at least one liter of crystalloid or equivalent amounts of colloid solution have been demonstrated by four recent studies.[10–13] For example, Collins and his associates[11] determined maternal blood pressure in 104 gravidae receiving epidural analgesia for labor. All were in a left lateral recumbent position. Fifty-one were hydrated with one liter of electrolyte solution shortly before institution of the block, and the incidence of arterial hypotension was 2%. In contrast, in the 53 women who were not preloaded, the incidence was 28%. Huovinen and his group[12] measured maternal arterial pressure and placental intervillous blood flow in 38 parturients about to undergo elective cesarean section. Eleven of 24 women who elected epidural analgesia were prehydrated with 10 ml/kg of a plasma expander (Haemaccel®) within 10 minutes of injection of the local anesthetic, and 13 were not. The remaining 14 women who chose general anesthesia served as controls. In the group not receiving preloading, maternal blood pressure and intervillous blood flow decreased significantly after establishment of the epidural block ($p < 0.05$ and 0.01, respectively). In contrast, when forced hydration had been undertaken, intervillous blood flow remained in the range of the control group and maternal arterial pressure declined only slightly. Measurements of central venous pressure in 20 gravidae receiving two liters of crystalloid solution following an overnight fast before initiation of epidural analgesia for elective cesarean section showed a rise from a mean prehydration level of 4.05 + 0.5 cm H_2O to a mean of 5.72 + 0.5 cm H_2O following fluid loading and establishment of the block, thus confirming the safety of fluid preloading.[13]

Cardiac standstill. The central nervous system and cardiovascular effects of local anesthetic drugs have long been recognized as complications of overdosage or accidental intravascular administration but have, in general, been readily treatable. However, since the introduction of the long-acting anesthetic bupivacaine in 1973, the incidence of fatal cardiac arrest has been increasing following attempted epidural block for cesarean section. Typically, a short grand mal seizure has been followed by disappearance of arterial pressure and pulse. Electrocardiographic manifestations have ranged from ventricular fibrillation to complete heart block to asystole. The interval between appearance of central nervous

system symptoms and cardiac collapse has been shorter than with other local anesthetics, such as lidocaine. Resuscitation has been unusually difficult, requiring prolonged cardiac compression and often direct current countershock in addition to prompt endotracheal intubation and controlled ventilation with 100% oxygen.[14] External cardiac compression is most successfully achieved on a hard surface, i.e., resuscitation board. In a hypotensive pregnant woman, however, this position is associated with severe aortocaval compression. Therefore, prompt delivery of the infant or, if delivery is not feasible, expert manual displacement of the gravid uterus must be undertaken to assure adequate venous return to the heart.[15]

As with all anesthetic complications, prevention is the best cure. Precise definition of the epidural space in association with test doses sufficiently large to elicit early central nervous system signs and symptoms (e.g., jitteriness, tinnitus, metallic taste in the mouth) minimizes the likelihood of intravascular injection of cardiotoxic quantities. All maintenance doses must be fractionated with sufficient intervals between injections to permit manifestations of toxicity to develop.[16] Addition of epinephrine to the test dose will produce tachycardia and palpitations and facilitate recognition of intravascular administration. However, the addition of epinephrine to the maintenance dose of local anesthetic remains controversial; although advocated by some, others are concerned about the potential risk of decreased intervillous blood flow,[17] particularly if the fetus is already compromised.

In August 1983 a warning: "bupivacaine 0.75% is no longer recommended for obstetrical anesthesia" was sent by the manufacturers of the drug to all anesthesiologists and obstetricians as a "Dear Doctor Letter," endorsed by the FDA's Anesthetic and Life Support Drugs Advisory Committee in October, and promulgated by the FDA Drug Bulletin in November. During the five months subsequent to the "Dear Doctor Letter," there have been no new cases of bupivacaine-induced seizures and cardiac arrest—in contrast to 10 cases reported to the FDA over the previous two years.[18]

Mortality from General Anesthesia

Failed intubation. Failure successfully to intubate the trachea has become the leading cause of maternal mortality in both the United States and the United Kingdom. Although difficult intubation can usually be

managed without serious sequelae in a surgical patient, the situation is compounded in a pregnant woman by a more rapidly declining oxygen reserve (secondary to increased oxygen consumption and decreased functional residual capacity)[19] and a significantly enhanced risk of pulmonary aspiration of gastric contents (see below). As with all candidates for general anesthesia, careful review of the general physiognomy and oral cavity should alert the anesthesiologist to potential difficulty in intubation. When such difficulty is anticipated, regional analgesia becomes the method of choice. If there are contraindications to regional techniques, skillful awake intubation following topical anesthesia of the upper airway should be undertaken.[20] In a pregnant woman there are two additional potential problems regarding endotracheal intubation. First, edema of the laryngeal mucosa which develops in all gravidae may become clinically significant in preeclamptic patients or following prolonged strenuous bearing-down efforts prohibiting passage of the usual size endotracheal tube despite visualization of the larynx. This difficulty is generally overcome by choosing a tube 0.5 to 1.0 cm smaller in diameter than usual.[21] The second problem is inability properly to manipulate the laryngoscope handle because of large breasts which fall back against the neck. This difficulty is resolved by either taping the breasts to the side or by using a special short-handled laryngoscope.

Despite the most careful evaluations and precautions, there have been and will be gravid patients in whom intubation proves impossible after induction of general anesthesia. Unless the anticipated operation is lifesaving for the patient herself, she must be allowed to regain consciousness and spontaneous respiration while being oxygenated with maintenance of cricoid pressure to minimize the risk of aspiration. Either regional analgesia or awake intubation must be substituted. Because of the difficulty in anticipating intubation problems, surgical operation should not start until proper placement of the tube has been confirmed. Although auscultation of the lungs is mandatory, "breath" sounds may be transmitted from the stomach following placement of the tube in the esophagus. Therefore, a capnograph or a device such as the "Einstein carbon dioxide detector"[22] should be available to ascertain intubation into the trachea with definity. Furthermore, each delivery room should be equipped with means of performing emergency cricothyroidotomy and transtracheal ventilation.

Pulmonary aspiration of gastric contents. Formerly the most frequent

etiology of anesthesia-related maternal death, pulmonary aspiration of gastric contents has become the second leading cause. Various anatomic, physiologic, and pharmacologic phenomena increase the risk of aspiration in the gravida. Anatomically, there is a progressive shift from the vertical to the horizontal axis of the stomach secondary to upward pressure by the enlarging uterus. This results in an increased intragastric pressure which is accentuated when the woman is placed in the lithotomy position. Physiologically, on a hormonal basis, gastric tone and motility are reduced. The situation is compounded by an above-normal gastric secretion (including hydrochloric acid) and by relative incompetence of the gastroesophageal sphincter. Finally, pain, anxiety, and drugs commonly administered during labor (e.g., narcotics, scopolamine) further delay gastric emptying.[23-27] Thus, no parturient presenting for anesthesia may be considered to have an "empty stomach."

Pulmonary aspiration of gastric contents may result from either active vomiting or passive regurgitation. In either case, the ensuing clinical situation depends on the nature of the aspirate. Inhalation of liquid gastric contents with a pH of 2.5 or lower leads to the acid aspiration syndrome first described by Curtis Mendelson and bearing his name.[28] Irritation of bronchial mucosa by acidic liquid causes bronchiolar spasms, peribronchiolar exudates, focal hemorrhages, and areas of necrosis. Aspiration of solid food particles, in contrast, results in varying degrees of airway collapse and reflex bronchospasm. Significant obstruction of the large airways or flooding of both lungs with acidic fluid may lead to a spectrum of clinical entities ranging from pneumonitis to hypoxic cardiac arrest.

Preventive measures begin with teaching pregnant women to avoid food and drink (other than clear fluid) when labor is imminent. Following admission to hospital, appropriate intravenous therapy avoids dehydration and starvation ketosis. Ingestion of an antacid will lower the acidity and raise the pH of gastric juice to safe levels (above 2.5).[29] Because the stomach divides into two pouches during recumbency, the gravida should turn from side to side to insure adequate mixing of the antacid with the gastric contents.[30] Although H_2-blocking drugs such as cimetidine will minimize gastric secretion, they fail to neutralize the acid already present.

At the time of induction of anesthesia, pretreatment with a small dose of a nondepolarizing neuromuscular relaxant will prevent succinylcholine-induced fasciculations which increase intragastric pressure.[31] From the

moment the patient begins to lose consciousness, expert cricoid pressure (Sellick's maneuver) must be applied.[32] This maneuver consists of constant pressure of the cricoid, the only complete tracheal ring, onto the esophagus, thereby effectively sealing the esophagus until such time as the endotracheal tube cuff has been inflated and the proper location of the tube confirmed. A recent editorial stated that "gastric aspiration into the lungs during anesthesia is preventable if a trained anaesthetist has the assistance of a trained assistant and has proper equipment."[33]

When inhalation or ketamine analgesia is provided for uncomplicated vaginal delivery, care must be taken to remain in constant communication with the patient, thus insuring a level of consciousness adequate to maintain the protective laryngeal reflexes.

Conclusion

The decrease in anesthesia-related maternal mortality noted over the last four decades is mainly the result of an enhanced awareness of the physiologic and pharmacologic differences between surgical and obstetric patients. Provision of obstetric anesthesia around the clock by subspecialists who keep abreast of new developments in the field will further improve the safety of anesthesia, not only for the mother but also for the baby.

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